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## Ophthalmology Lecture 4: Glaucoma for the Family Physician

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# GLAUCOMA FOR THE FAMILY PHYSICIAN

ELWA Family Medicine Residency Program

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BONO REGION, GHANA

# GOAL

- To appreciate glaucoma as a sight-threatening condition.
- Recognize the risk factors of glaucoma.
- to provide a background to glaucoma, and describe the assessment and management of glaucoma patients.
- finally to equip the Family Physician to be able to diagnose glaucoma, and refer appropriately

# OUTLINE

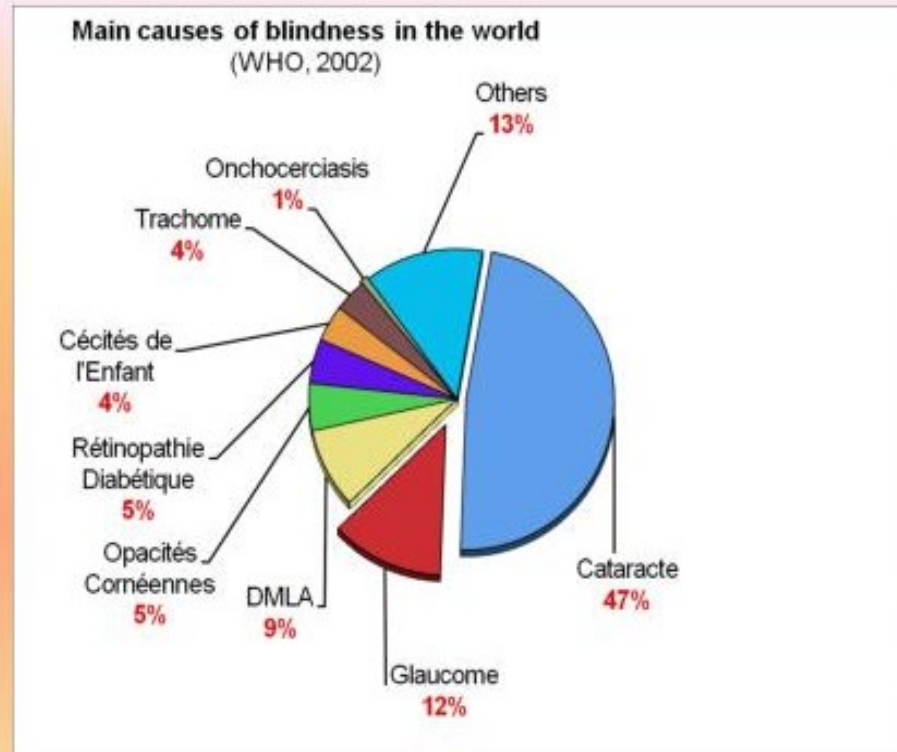
- INTRODUCTION
- EPIDEMIOLOGY
- CLASSIFICATION
- SOME COMMON GLAUCOMA TYPES
- MANAGEMENT
- CONCLUSION

# GLAUCOMA

- A group of diseases characterized by progressive optic neuropathy with corresponding visual field defects where IOP is a major risk factor
- Glaucoma causes irreversible damage to the optic nerve and no known treatment will restore lost vision.
- According to The World Health Organization, although 70 million people worldwide suffer from glaucoma, it is treatable if detected early, which means that those suffering from the disease can preserve their vision.

- WHO: The second biggest cause of blindness and visual impairment globally
- No.1 cause of irreversible blindness in most African populations

## Causes Of World Blindness



### Avoidable

- ✓ Trachoma
- ✓ Onchocerciasis
- ✓ Corneal opacities

### Avoidable/Treatable

- ✓ Cataract
- ✓ Congen. Cataract & Glaucoma

### Still difficult to manage +++

- ✓ Glaucome
- ✓ D. Retinopathies

# The Global Burden of Glaucoma

- A conservative estimation of the prevalence of glaucoma in Africa is 4% of people in the age group > 40 years
- >50% unaware of disease in developed world
- >90% unaware in developing countries

Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. *Br J Ophthalmol.* 2006;90:262–267

# Glaucoma Burden in Africa

## -Blindness-

- Estimated to account for 4.4% of blindness in Africa

Naidoo K, Gichuhi S, Basáñez MG, et al. Prevalence and causes of vision loss in sub-Saharan Africa: 1990-2010. *Br J Ophthalmol*. 2014;98:612-61

- Nigeria-16.7% of total blindness burden

The Nigerian Blindness and visual impairment survey

- In Ghana, it accounts for 19.4% of all causes of blindness

Ghana Blindness and visual impairment survey



# Glaucoma-classification

- Anatomic-Open angle or closed angle
- Presentation-Chronic or acute
- Cause-Primary or secondary

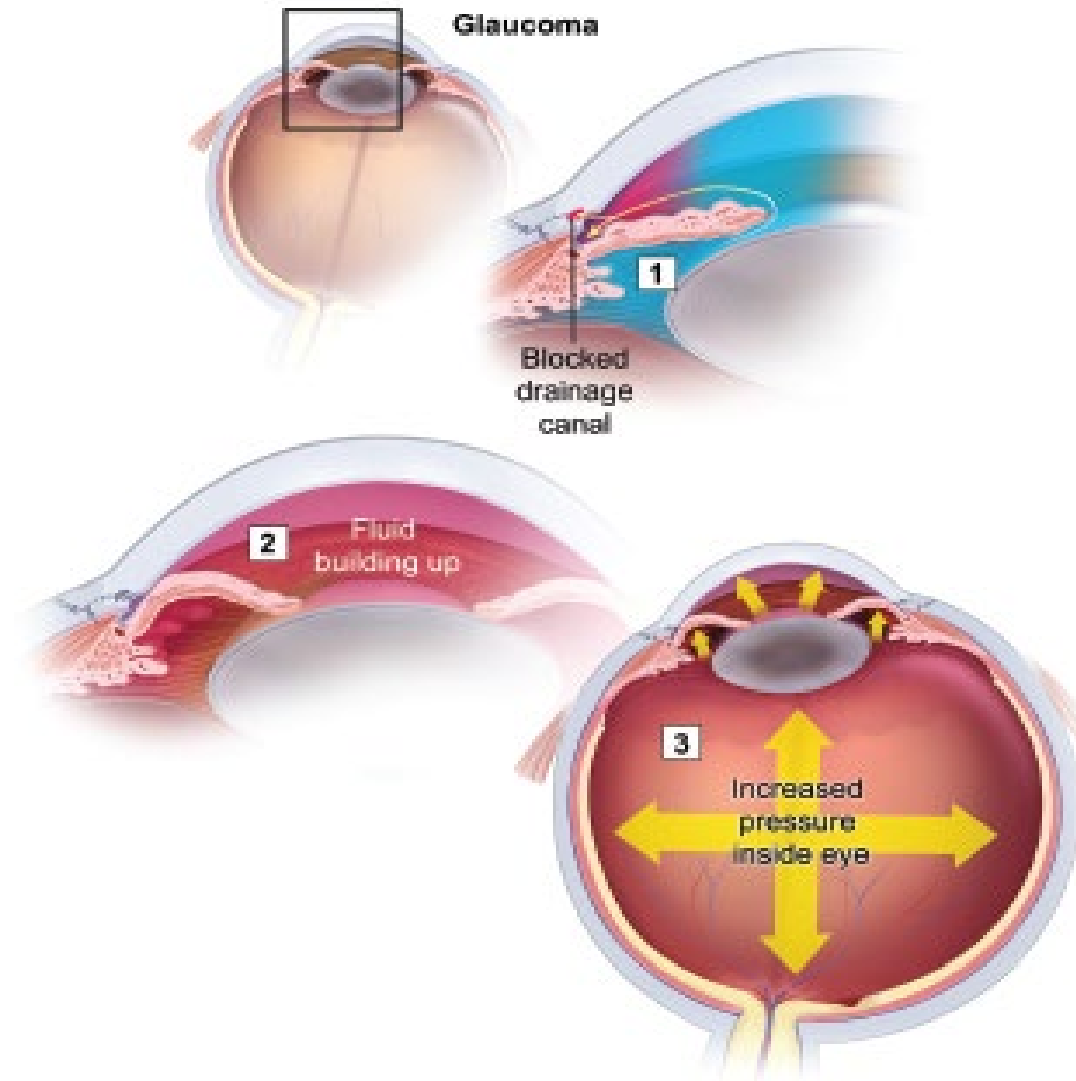
# Classification of glaucoma

1. Primary open angle glaucoma (POAG)
  2. Primary angle closure glaucoma (PACG)
  3. Secondary glaucoma
  4. Congenital glaucoma (CG)
- The most common type of glaucoma is primary open angle glaucoma also called chronic open angle glaucoma or chronic simple glaucoma and

## Aqueous humour (aqueous)

- Aqueous is produced in the ciliary body
- It circulates around, and provides nourishment for, the lens
- It passes through pupil into the anterior chamber
- It leaves eye via the trabecular meshwork in the angle and enters the episcleral veins

# GLAUCOMA

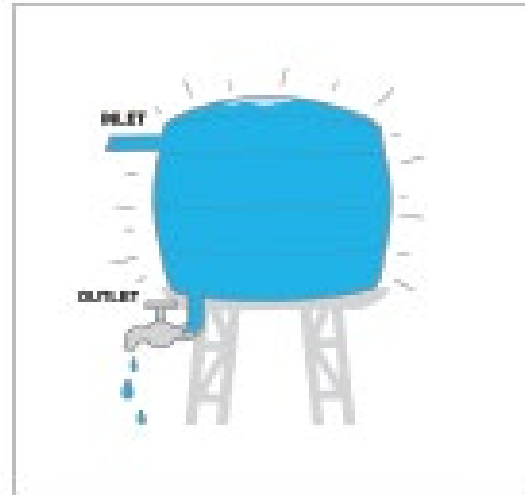


# Intraocular pressure (IOP)

- IOP is a balance between aqueous production by the ciliary body and aqueous drainage through the angle
- If aqueous drainage is impaired, the IOP rises
- Normal IOP is below 21 mm Hg (defined as 2 standard deviations above the mean)



Normal

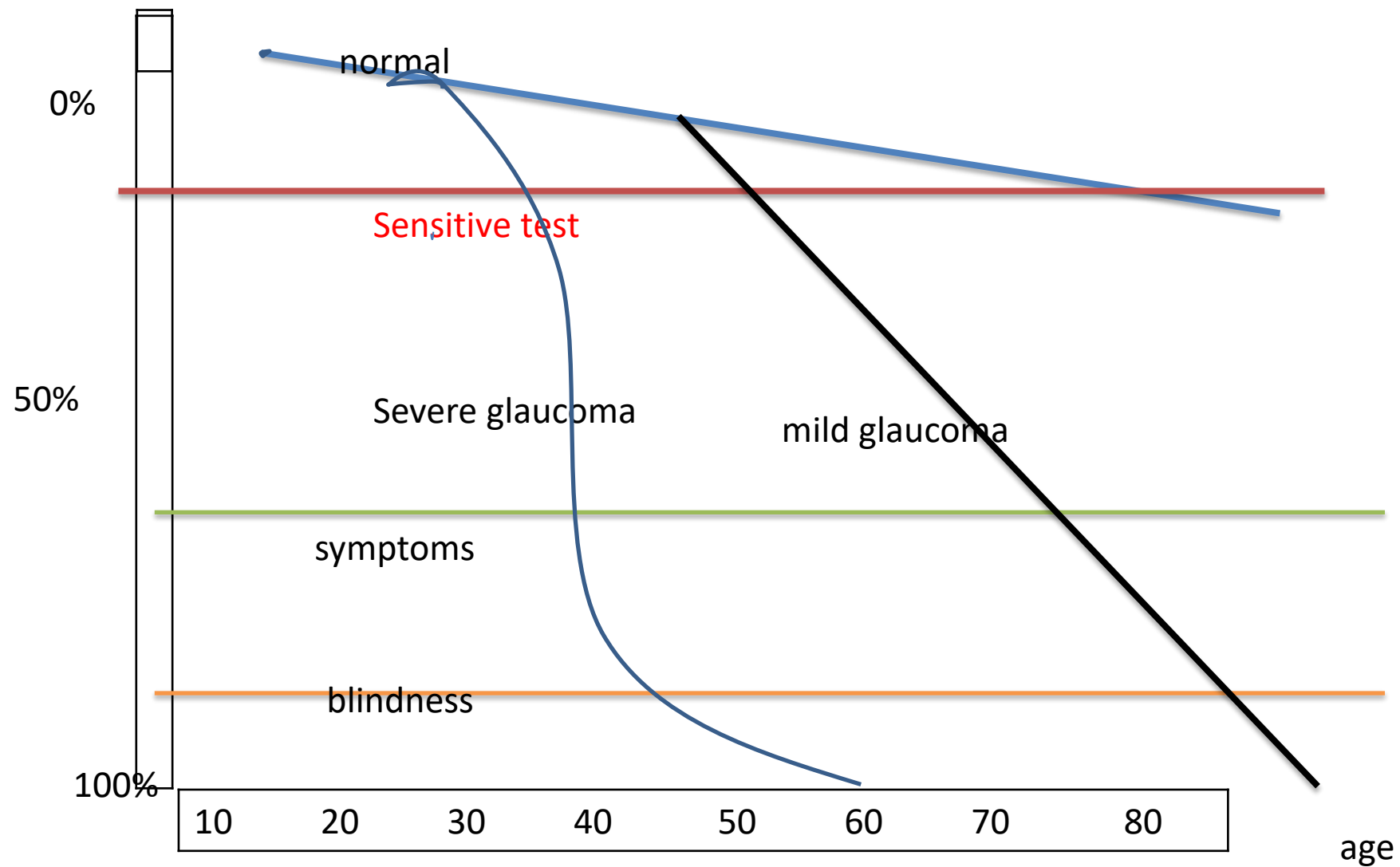


Affected



Treated

# Loss of neurones in optic nerve with age and disease



## Glaucoma - effect on visual field



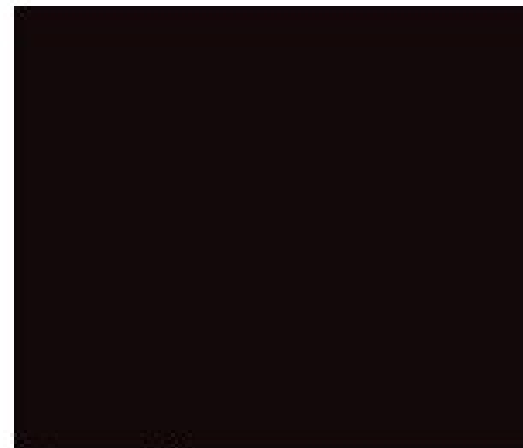
Normal



Early Glaucoma



Advance Glaucoma



Extreme Glaucoma



## PRIMARY OPEN ANGLE GLAUCOMA (POAG)

- POAG is a chronic progressive optic neuropathy with gradual loss of nerve fibres
- 80% of patients have IOP > 21mmHg
- Enlargement of optic disc cup (loss of neurones)
- Progressive loss of visual field (tunnel vision)

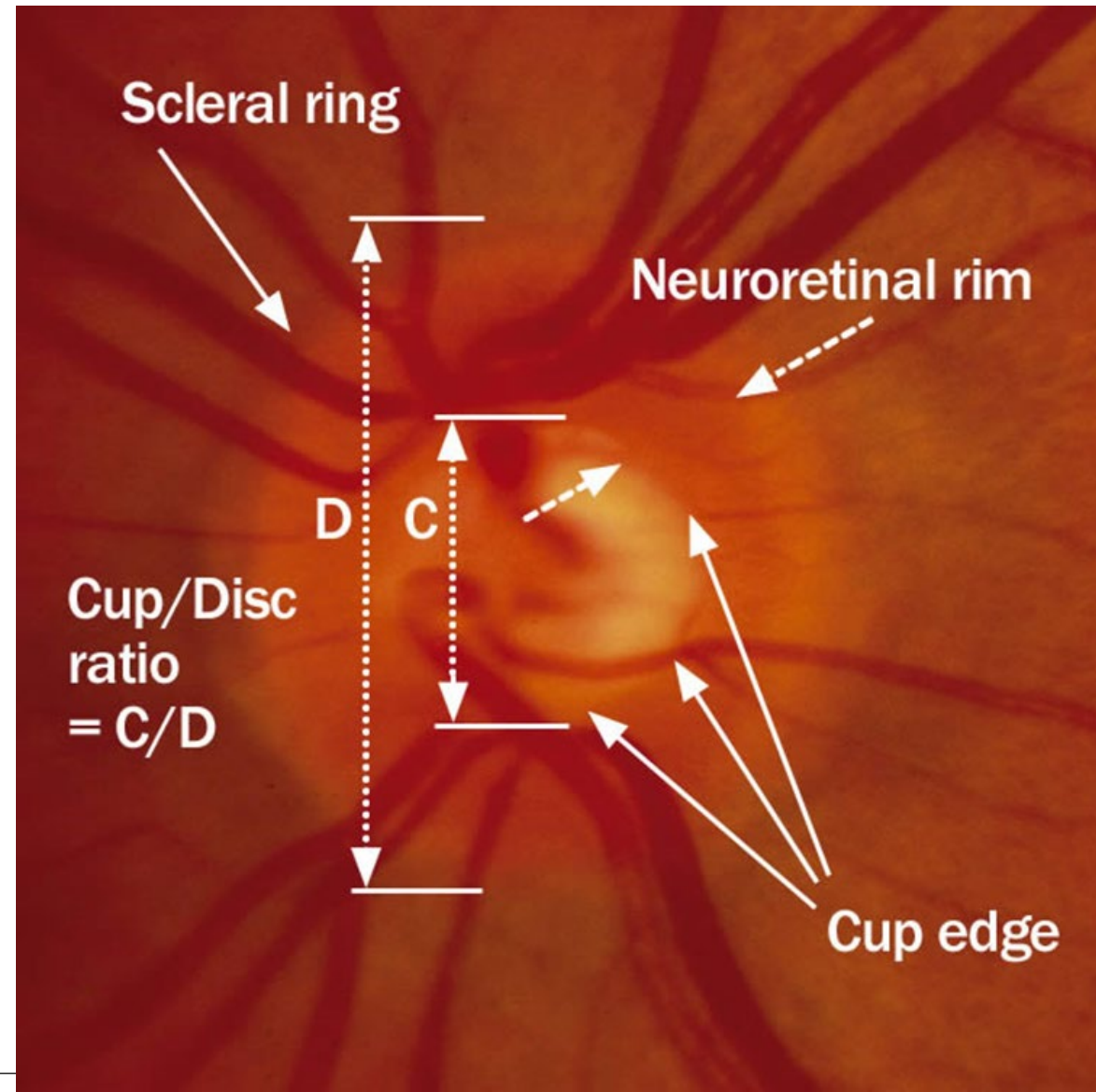
# POAG Symptoms

- Many asymptomatic “thief of sight”
- Bilateral but often asymmetric at time of Dx
- Early peripheral vision loss
- Late central vision loss
- Decreased contrast sensitivity
- Diminished color vision

# Risk factors

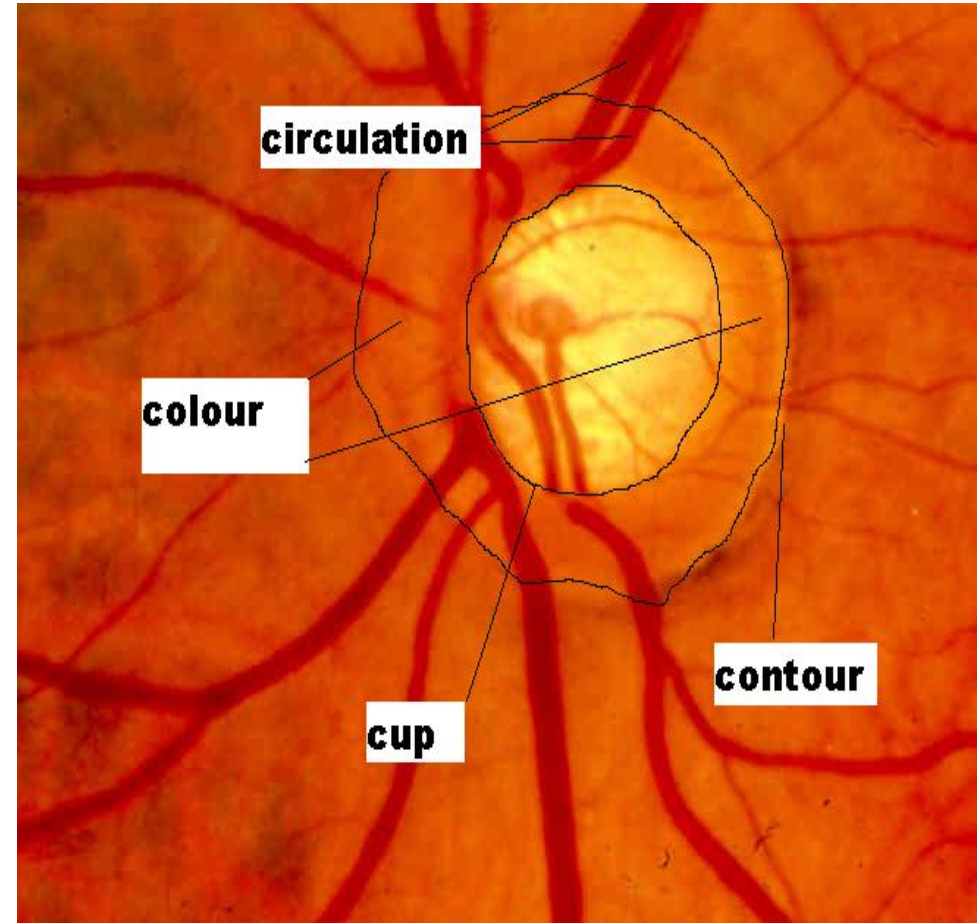
- Race: African & Latino
  - Africans have higher mean IOP, larger c:d ratio, ↑ prevalence of POAG, earlier onset, 8x risk of blindness
- Age
  - IOP increases with age
- FH
  - 1<sup>st</sup> degree relatives at highest risk (10% risk of developing POAG)
  - 25% of new POAG cases have + FH
- DM (mixed results)
- POAG/OHT is 2X - 3X more prevalent in DM
- DM or +GTT is more prevalent in POAG or steroid responders
- Myopia
  - Same mean IOP as hyperopes but 3X POAG
- Perfusion pressure =  $SBP - IOP$
- Steroids
- Trauma

# NORMAL OPTIC DISC



# Optic disc cupping

- Most optic discs have a central physiological cup which usually has a diameter less than half of the disc diameter
- The cup is the part of the disc without nerve fibres and blood vessels
- Small optic discs have small cups and still be healthy
- Large optic discs can have large cups and still be healthy



### Features of glaucomatous disc

- Bayonetting of blood vessel
- Cup-to-disc ratio of more than 0.4
- Pale disc with nasally displaced blood vessels
- Thin rim that does not comply with the ISNT rule
- Peripapillary choroidal atrophy

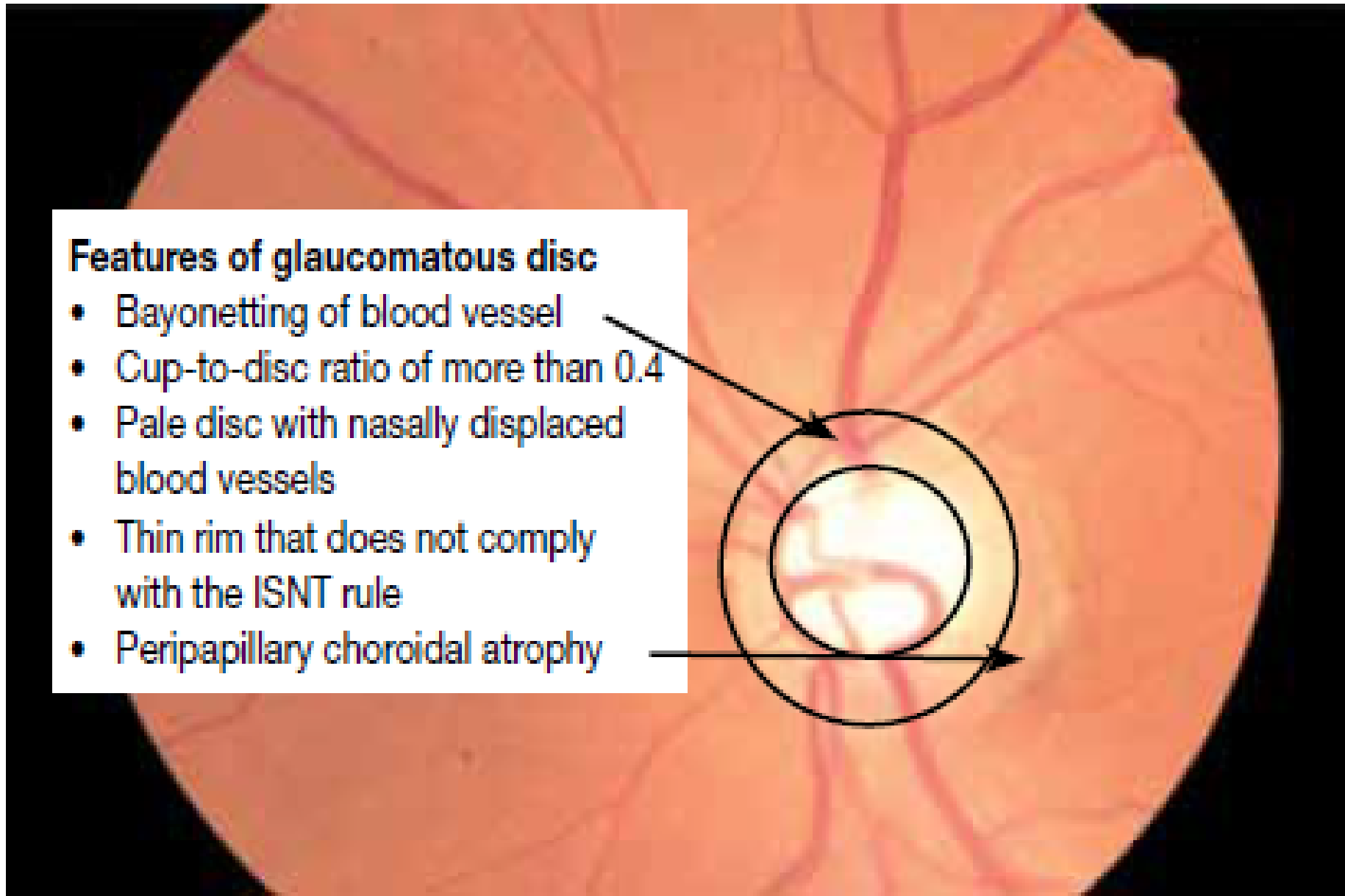
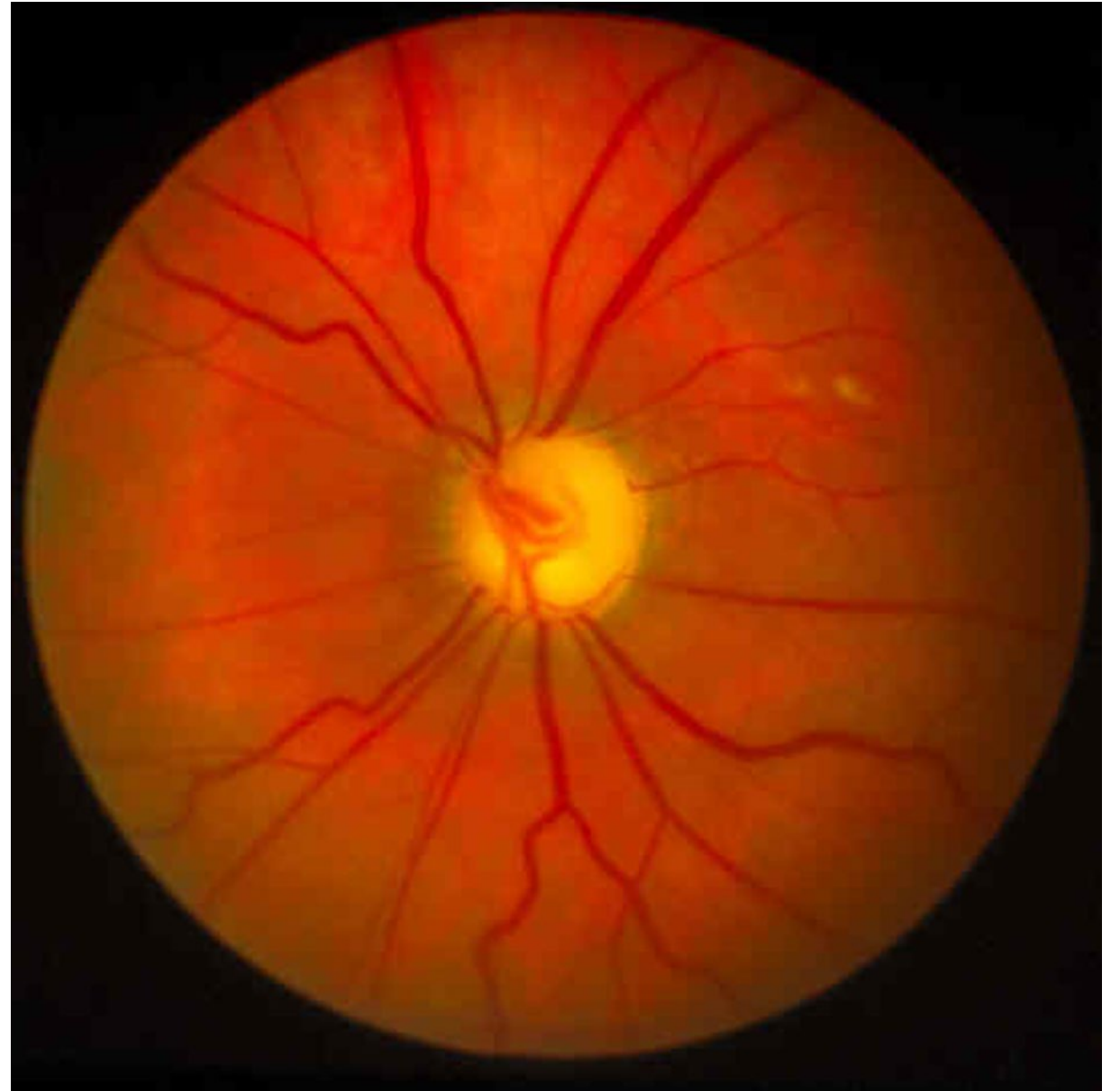
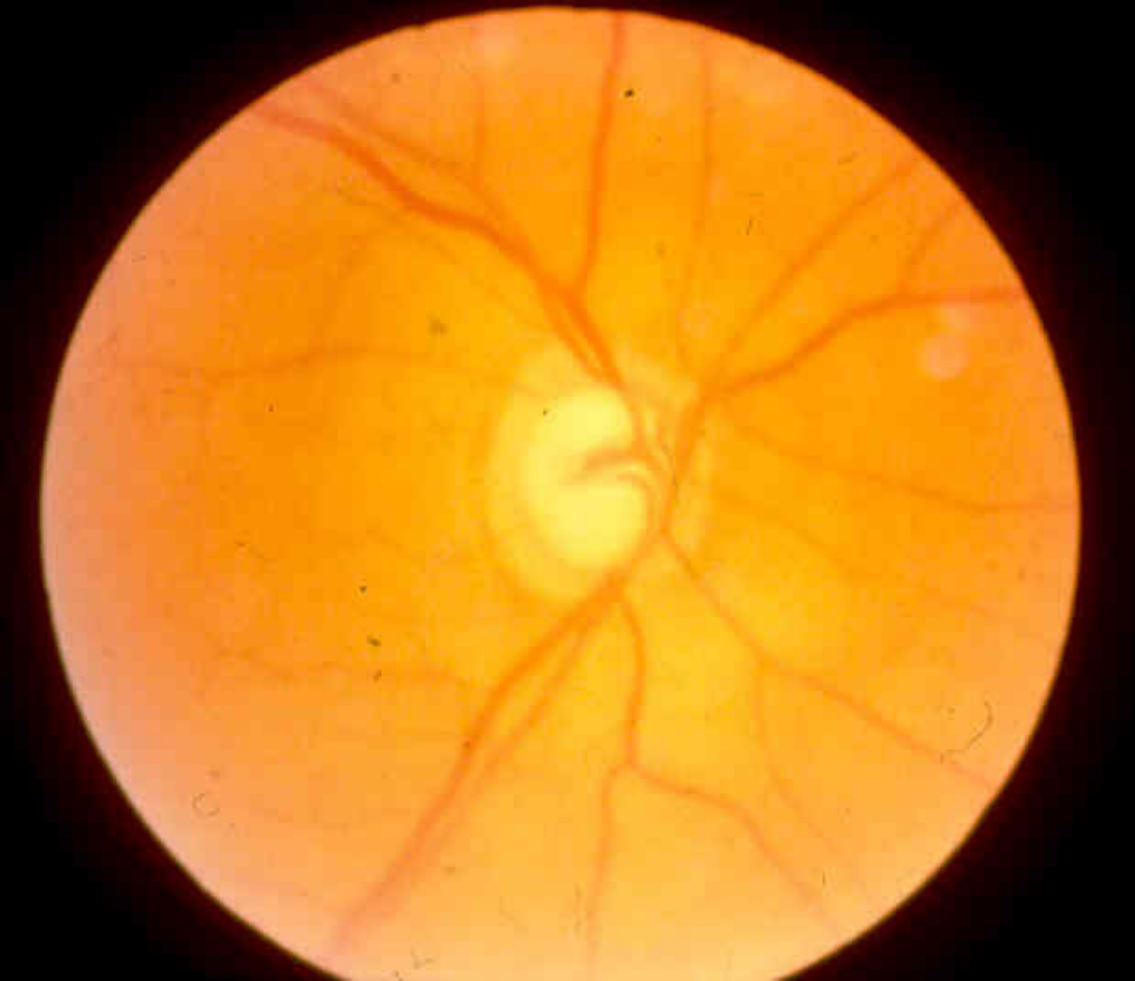


Figure 3: Photograph showing features of a glaucomatous optic disc

# GLAUCOMA



# Diagnosis of glaucoma

- Diagnosis of glaucoma depends on the presence of the following three components:
- Raised IOP (normal IOP is 10–21 mmHg).
- Structural damage to the optic nerve head (optic disc).
- Loss of visual field in a characteristic way.

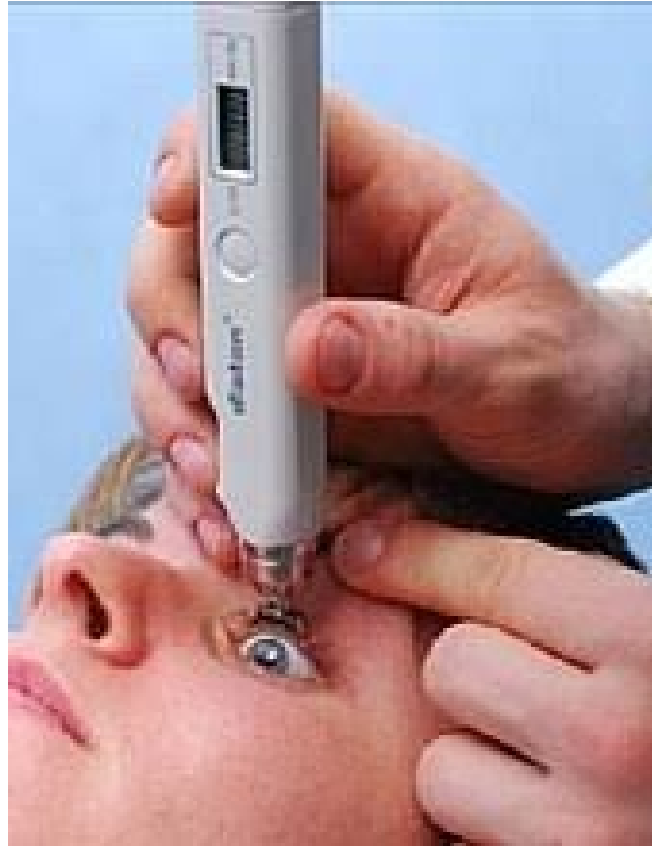


# Assessment of patients with glaucoma

- Assessment includes the measurement of the IOP, clinical evaluation of the optic disc, and a visual field evaluation.
- Different instruments can be used to measure the IOP.
- The Goldmann applanation tonometer is the standard tonometer used by ophthalmologists and some optometrists.
- Most optometrists use an air-puff tonometer, Icare tonometer and tonopens

- An examination with a direct ophthalmoscope gives an excellent view of the optic disc.
- The use of fundoscopy and pupil reactions as screening tests for all persons at risk (age group > 40 years) have proved to be suitable screening tools for glaucoma.
- Imaging studies.... VISUAL FIELD TEST(VFT), OPTICAL COHERENCE TOMOGRAPHY(OCT), GDx

# TONOMETRY



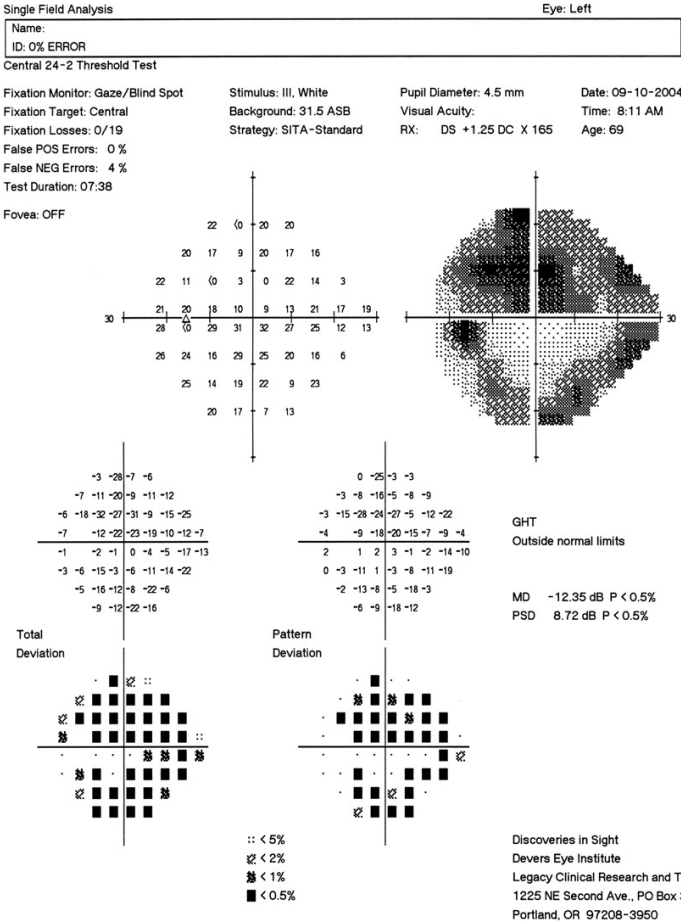


# Visual field defects

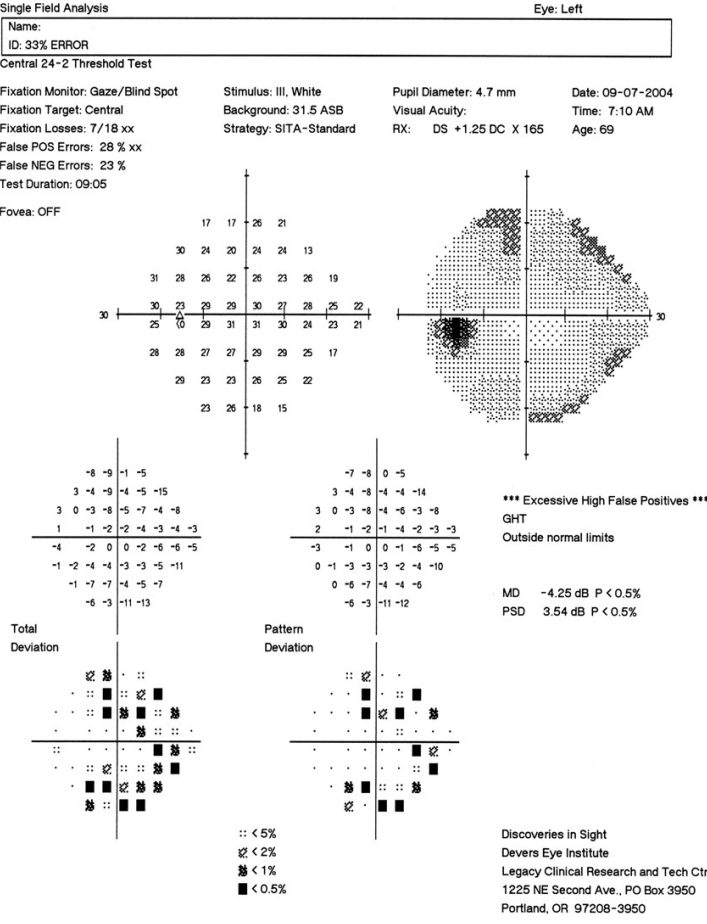
- Visual field defects are difficult to detect clinically without specialised equipment until late in the disease (loss of  $> 50\%$  of the nerve fibres).
- Arcuate scotomas begin in superior or inferior visual field
- These join up to produce a ring scotoma
- This extends to produce tunnel vision
- Eventually central vision is lost
- Visual acuity remains good until very late in the condition

# VISUAL FIELD TEST

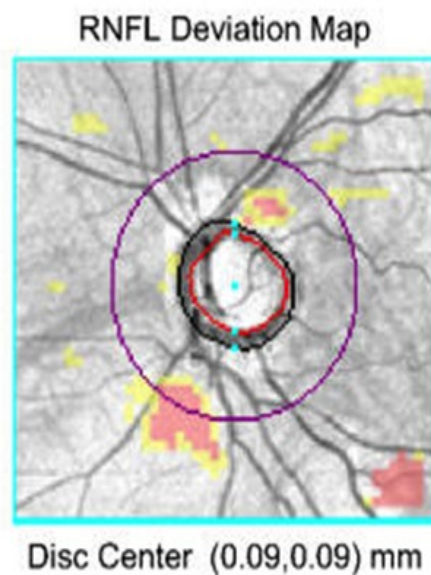
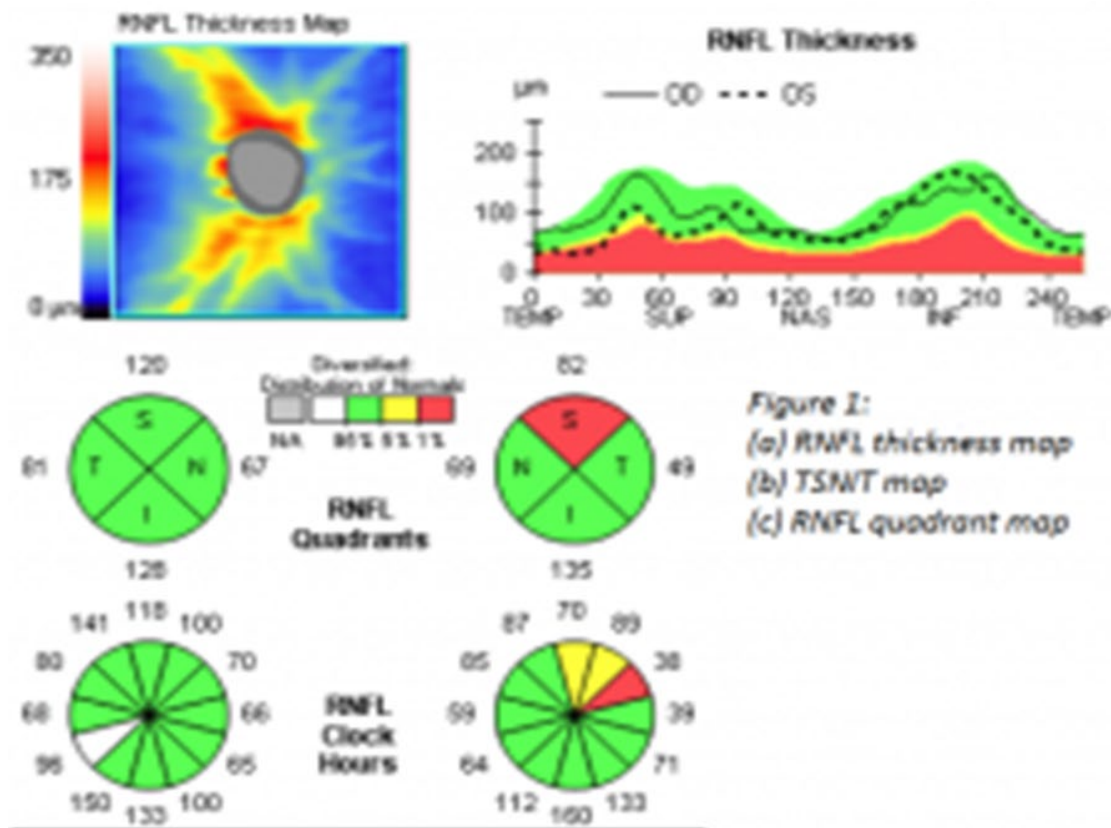
A



B



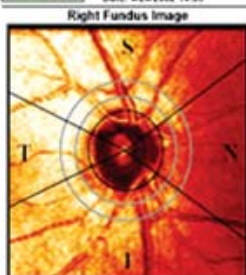
# OCT



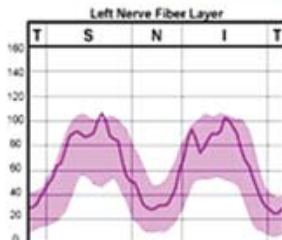
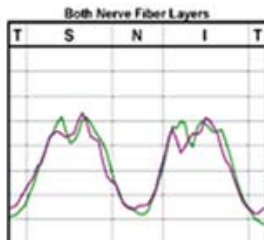
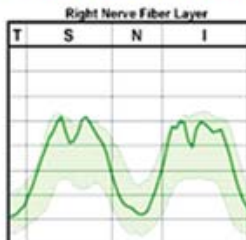
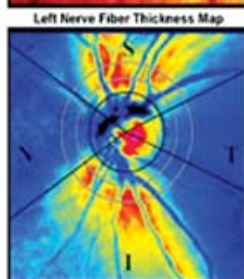
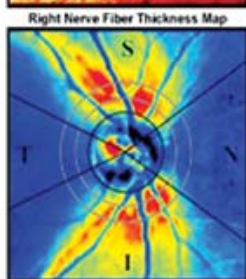
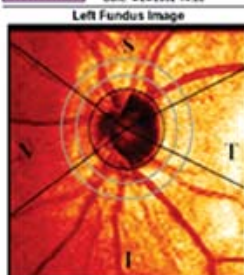
|                        | OD                  | OS                  |
|------------------------|---------------------|---------------------|
| Average RNFL Thickness | 77 $\mu\text{m}$    | 80 $\mu\text{m}$    |
| RNFL Symmetry          | 58%                 |                     |
| Rim Area               | 0.88 $\text{mm}^2$  | 0.77 $\text{mm}^2$  |
| Disc Area              | 1.97 $\text{mm}^2$  | 2.01 $\text{mm}^2$  |
| Average C/D Ratio      | 0.75                | 0.80                |
| Vertical C/D Ratio     | 0.80                | 0.78                |
| Cup Volume             | 0.472 $\text{mm}^3$ | 0.616 $\text{mm}^3$ |

Figure 2: Optic Nerve Head Analysis

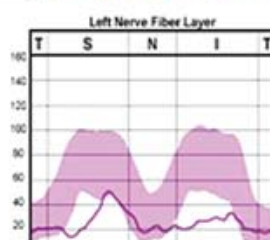
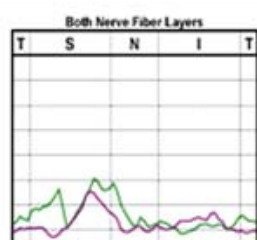
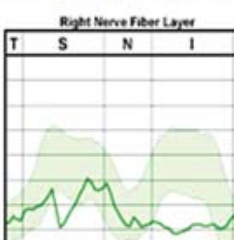
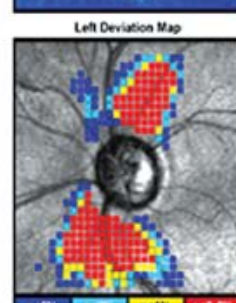
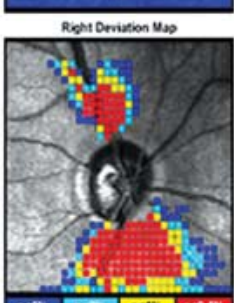
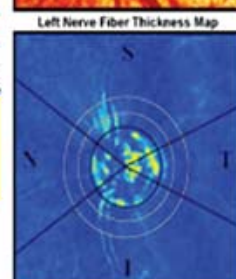
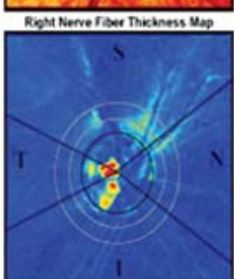
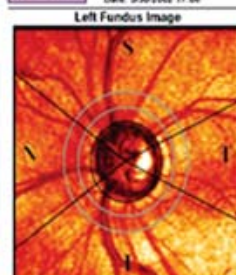




| TSNIT<br>Parameters | OD<br>Actual Val. | OS<br>Actual Val. |
|---------------------|-------------------|-------------------|
| TSNIT Average       | 64.13             | 63.62             |
| Superior Average    | 83.24             | 81.61             |
| Inferior Average    | 79.37             | 73.95             |
| TSNIT Std. Dev.     | 29.54             | 26.13             |
| Inter-Eye Symmetry  | 0.96              |                   |
| NFI                 | 4                 | 10                |



| TSNIT Parameters   | OD<br>Actual Val. | OS<br>Actual Val. |
|--------------------|-------------------|-------------------|
| TSNIT Average      | 32.23             | 25.79             |
| Superior Average   | 44.81             | 32.00             |
| Inferior Average   | 22.70             | 24.94             |
| TSNIT Std. Dev.    | 11.53             | 8.45              |
| Inter-Eye Symmetry | 0.50              |                   |
| NFI                | 88                | 98                |





## Aims of treatment of POAG

- To stabilise visual field loss
- To reduce risk of further progression
- To maintain central visual field
- Once lost, visual field cannot be recovered

# Management of glaucoma

- POAG can be managed by:
  - Observation: mainly ocular hypertension
  - Medical therapy: drops (rarely oral therapy)
  - Laser therapy: laser trabeculoplasty, ciliary body laser, laser iridotomy
  - Surgery: trabeculectomy

# Medical therapy

- Topical
  - Beta-blockers e.g. Timolol
  - Parasympathomimetic eg pilocarpine
  - Prostaglandin analogues e.g. xalatan
  - Carbonic anhydrase inhibitors e.g. dorzolamide
- Oral
  - Carbonic anhydrase inhibitors e.g. diamox

# Side effects if medical therapy

- Beta blockers can cause postural hypotension, bronchospasm and worsening of asthma, impotence, heart block and congestive cardiac failure.
- Topical allergy to the drug, or the preservative in the drops, can cause itching, swelling of eyelids and eczema of the periocular skin.
- Side effects of acetazolamide (Diamox) can include nausea, paraesthesia, electrolyte disturbances and renal stones.
- Prostaglandins can cause an increase in melanin pigmentation in the iris, blurred vision, redness of the eyelids and anterior uveitis, upper respiratory tract infection symptoms, chest pain and miscarriages.

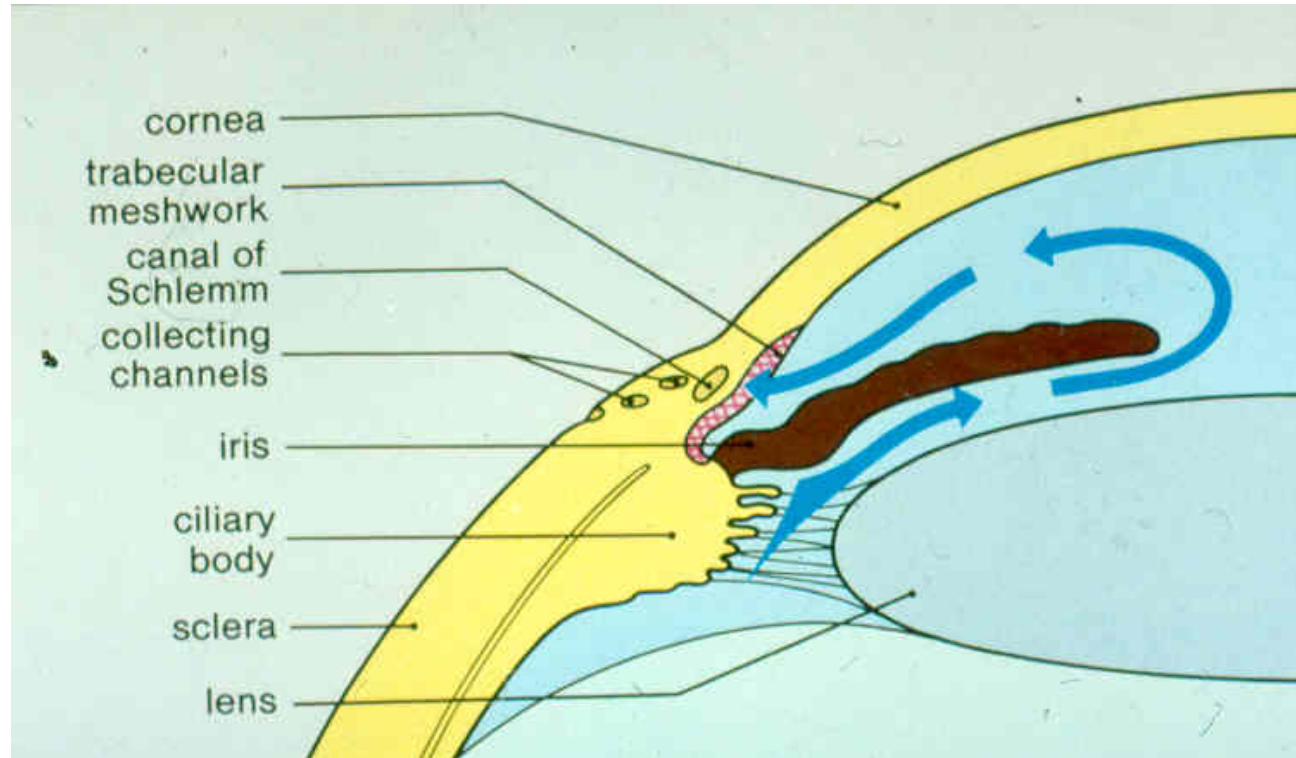
# Angle closure glaucoma

- Primary angle closure glaucoma (ACG) is caused by appositional or synechial closure of the anterior chamber angle, due to a number of mechanisms.
- This is a sight threatening emergency, involving painful loss of vision, due to sudden and total closure of angle.
- It is probably the best known type of glaucoma.

# ANGLE CLOSURE GLAUCOMA

- In ACG, apposition of the lens to the back of the iris prevents the flow of aqueous humour from the posterior chamber to the anterior chamber.
- This is more likely to occur when the pupil is semi-dilated at night.
- Aqueous humour that collects behind the iris pushes the iris onto the trabecular meshwork, preventing the drainage of the aqueous humour from the eye, and resulting in a rapid increase in IOP.

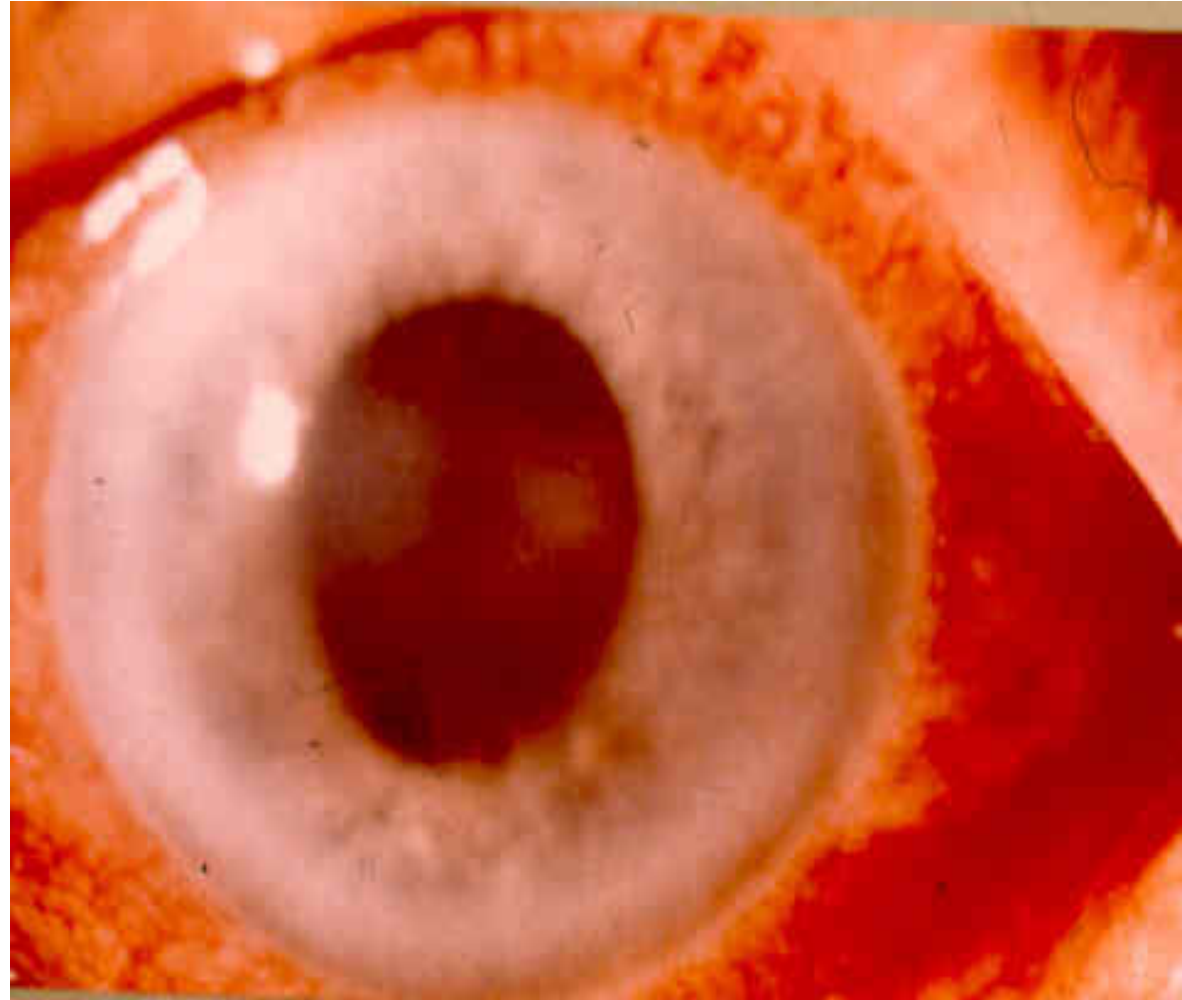
## Acute angle closure glaucoma: mechanism



- Aqueous is produced in the ciliary body
- It circulates around the lens and through the pupil into the anterior chamber
- It leaves the eye at the angle
- In some eyes, when the pupil is mid-dilated, aqueous cannot pass through the pupil
- The iris is pushed against the cornea and the angle closes
- This leads to a rapid build up of pressure, hence the severe pain

## Acute angle closure glaucoma: signs

- Very red eye
- Corneal oedema
- Mid-dilated pupil
- Poor vision





# Symptoms of ACG

- Symptoms of an acute attack are a painful red eye, headache and, frequently, nausea and vomiting.
- Vision is blurred, because the cornea becomes oedematous.
- There may be a history of similar attacks in the past that were aborted by going to sleep, because the pupil constricts and may pull the peripheral iris out of the angle, ending the attack.

# *Signs*

- Impaired visual acuity
- Red and painful eye
- Cornea is hazy, because of the oedema
- Pupil is semi-dilated and fixed, with no reaction to light
- On palpation, the affected eye feels harder than the other eye
- If the patient is seen shortly after an attack has resolved, none of these signs may be present. Therefore it is important to take a good history.

# Groups at risk of developing acute ACG

- Hypermetropic (far-sighted) patients
- Women (3–4 times higher)
- Eskimos (40 times higher) and Asians have a higher incidence than Caucasians
- Black patients have a low risk for developing ACG
- Highest incidence: 55–65 years of age

# Medical treatment for the acute attack

- Acetazolamide (Diamox<sup>®</sup>) 500 mg IV or per os
- Hyperosmotic agents, e.g. glycerine or mannitol
- Topical miotics, e.g. pilocarpine 2%, to constrict the pupil and pull the iris from the trabecular meshwork
- Analgesics
- Antiemetics
- Lie supine for one hour so that the effect of gravity can pull the iris from the angle.

# Surgery

- Surgery is the treatment of choice for ACG and is performed as soon as the cornea has cleared after the acute attack.
- A peripheral iridectomy or laser iridotomy can be performed.
- Both eyes are treated, because the risk of angle closure in the other eye is significant.
- Prophylactic treatment: Pilocarpine drops can prevent an acute attack until the patient can be referred for surgery or laser treatment.

# Secondary glaucoma

- In secondary glaucoma elevated IOP results in progressive optic disc neuropathy and visual field defects.
- The condition is caused by other ophthalmological or extraocular diseases and certain drugs.
- The FP must be aware of the underlying pathology that may cause secondary glaucoma, in order to prevent or delay the progression of glaucoma.

# common causes of secondary open angle glaucoma

- Treatment with steroids (topical and systemic, asthma inhalers, nasal sprays, and even topical ointments)
- Particles that block the trabecular meshwork (malignant cells, red blood cells, inflammatory cells or pigment)
- Membranes in the anterior chamber angle
- Trauma to the trabecular meshwork
- Neovascularisation in the angle, e.g. in diabetics, or after central retinal vein occlusion
- Pseudoexfoliation syndrome

# MANAGEMENT OF SECONDARY GLAUCOMA

- ADDRESS UNDERLYING CASE
- MEDICAL OR SURGICAL MEASURES TO REDUCE IOP



# Congenital glaucoma

- In congenital glaucoma (CG), a developmental malformation of the anterior chamber angle causes glaucoma.
- CG calls for early assessment and surgical intervention to prevent structural damage to the optic disc and allow visual development.
- CG is frequently bilateral and associated with other defects. It needs early diagnosis to avoid irreversible blindness.

# CONGENITAL GLAUCOMA

- **TRIAD OF SYMPTOMS**

1. EPIPHORA/LACRIMATION
2. PHOTOPHOBIA
3. BLEPHAROSPASM

## **SIGNS**

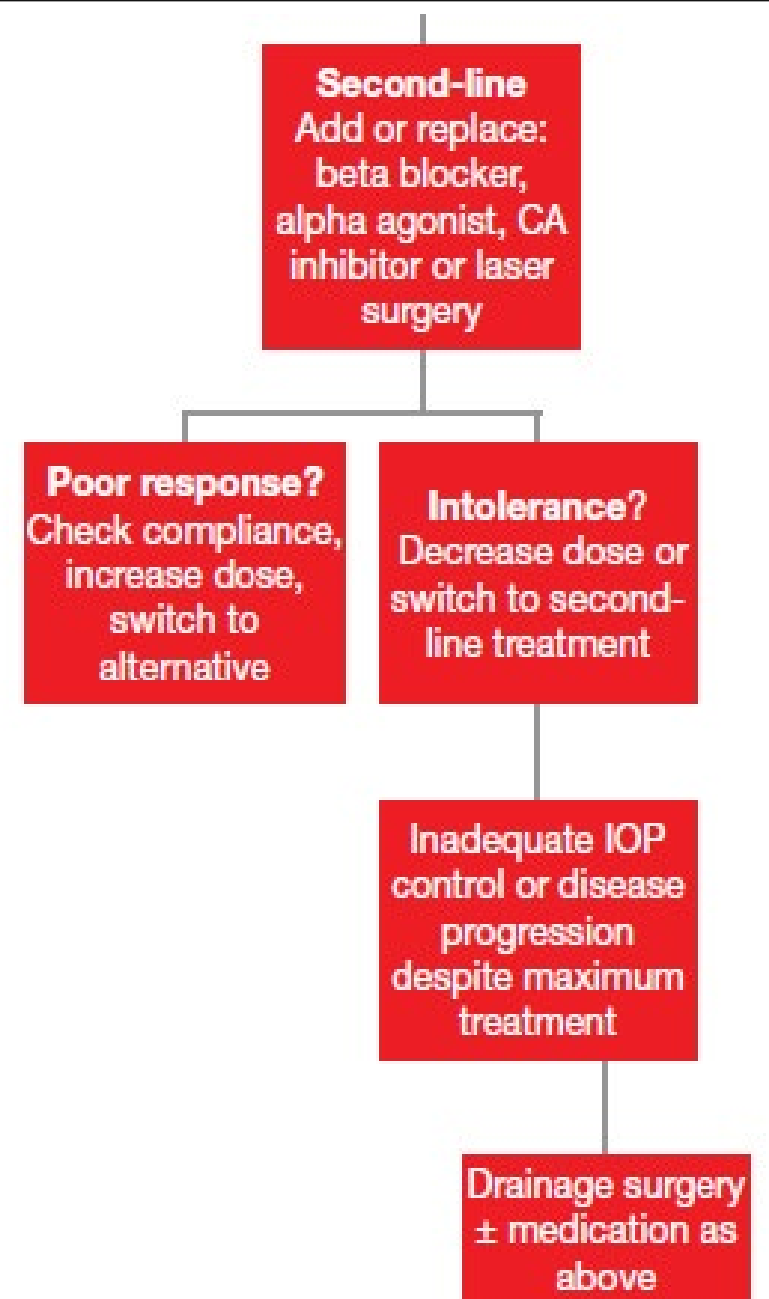
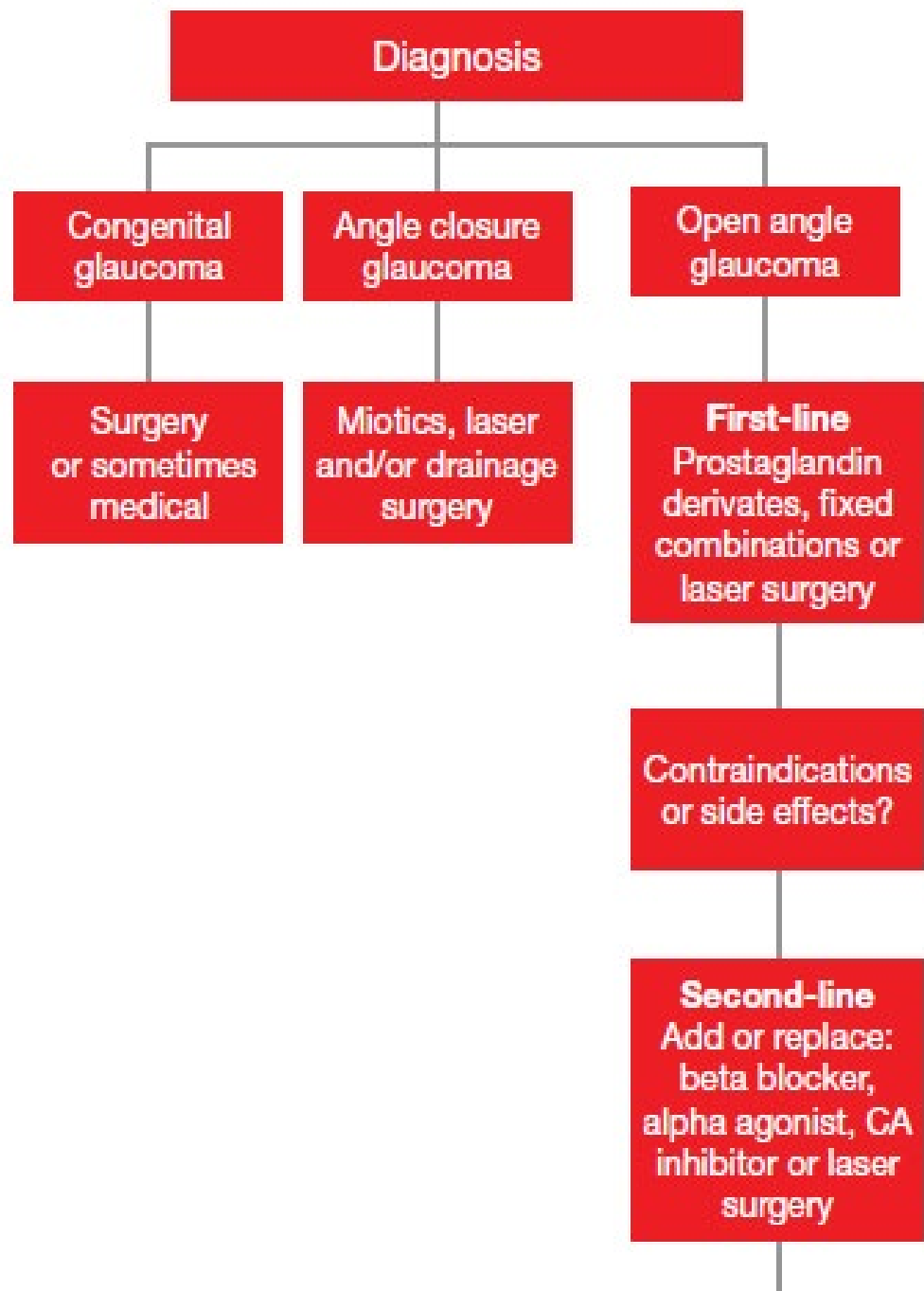
- corneal haze
- corneal opacity
- increased corneal diameter (> 12 mm), increased size of the eye or buphthalmos (due to raised IOP and elastic sclera)
- pale optic disc.



The eyes look big, with the corneas blurry and gray.

# Treatment

- Treatment involves surgery (goniotomy or trabeculotomy)



# Conclusion

- Identify patients with signs and symptoms suggestive of acute, angle closure glaucoma and institute management, in addition to referral to the ophthalmologist.
- Engage in screening of patients with symptoms and signs suggestive of open angle glaucoma.
- Explain and reassure the patient, because this is a chronic condition.
- discuss the impact of the visual loss with patients and their families i.e driving cessation and limitation, bumping into objects and falls.

# Conclusion

- Refer for reassessment if the patient has symptoms of progression or side effects from the treatment.
- Educate patients about screening of family members and the genetic factors in glaucoma. (First degree relatives have a 10% chance of developing glaucoma.)
- Refer a patient who has had previous glaucoma surgery (even years before), and who presents with a red eye or signs of infection, back to the ophthalmologist immediately, to exclude blebitis or endophthalmitis

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